SUMMARY OF RECENT INFORMATION REGARDING EFFECTS OF PCB's ON BIRDS AND MAMMALS

Rey C. Stendell*

Abstract

The significance of PCB's to wild animals depends both upon their lethal toxicity and their sublethal physiological effects. The lethal dietary toxicities of most Aroclors to experimental birds generally are less than those of DDE, DDT, or dieldrin. Effects of PCB's on reproduction are apt to have the most serious impact on populations. These effects are difficult to evaluate. The chief reproductive effects in chickens are reduced egg production and hatchability. Deformities in chicks are common, and growth rates of young sometimes are depressed. Administration of low dietary levels of PCB's to chickens, bobwhite quail, ringdoves, and mallards have not resulted in thinning of eggshells. No tests of shell thinning have been made with birds of prey or other critical species. Residues of PCB's in wild birds often are at levels known to have caused reproductive impairment in chickens. Species differences must be considered, however, and the significance of these levels to wild birds is very uncertain.

PCB's have been implicated in the reproductive failure and mortality of mink from the Great Lakes region. High levels of PCB's and organochlorine pesticides have been found in marine mammals and have been linked with premature births in some species. Recent studies have shown that some species of bats may be sensitive to a low level of PCB contamination.

INTRODUCTION

The widespread distribution of PCB's in the environment and their effects upon populations of wild birds and mammals are matters of growing concern. Their presence has stimulated research to evaluate their role in the biosphere. This paper reviews recent literature (1972-1975) on the effects of PCB's upon birds and mammals.

EFFECTS ON BIRDS

Lethal Toxicity

Outright mortality can affect populations. Measurement of direct toxicity is an important first step in the evaluation of a chemical. Toxicities of different PCB's to young pheasants (*Phasianus colchicus*), mallards (*Anas platyrhynchos*), bobwhite quail (*Colinus virginianus*), and coturnix quail (*Coturnix coturnix*) have been compared with the toxicities of DDT, dieldrin, and other insecticides (ref. 1). LC_{50}'s for the PCB's were high. Tests of six PCB mixtures, containing 32 to 62 percent chlorine, showed that the toxicity increased with the percentage of chlorine. The dietary toxicities of the Aroclors were, with few exceptions, less than those of the four organochlorine pesticides used for comparison. Special tests with coturnix quail showed that the toxic effects of DDE and Aroclor 1254 were additive but not synergistic. In other studies, Aroclor 1254 was approximately as toxic as DDE to four species of blackbirds (ref. 2). Redwinged blackbirds (*Agelaius phoeniceus*) were somewhat more susceptible to DDE than to PCB's.

Regular oral doses from 10 to 210 mg Aroclor 1254 produced some mortality among subadult pheasants (ref. 3). The amount and timing of the mortality were related to the dose. Heavier birds lived longer and lost the greatest percentage of their body weight before death.

Residues of PCB's in brains of blackbirds given heavy doses of Aroclor 1254 were diagnostic of death (ref. 4); residues in brains of birds that died varied from 349 to 763 ppm (wet weight) and were not above 301 ppm in survivors. In studies with pheasants given daily doses of 210 mg of Aroclor 1254, a brain residue level of 300 to 400 ppm generally indicated death due to PCB toxicosis (ref. 3). Residues of PCB's in livers and other tissues were more variable and of lesser value for diagnosing death.

PCB residues in brains of bald eagles (*Haliaeetus leucocephalus*) found dead from various causes in the United States between 1969 and 1972 varied from 0.10 to 230 ppm (refs. 5,6). Even the highest levels in the eagle brains were below the lethal range determined with captive blackbirds. However, there may be at least two distinct modes of death from PCB's (ref. 7). A sudden heavy intake may cause death from neurotoxicity, with brain residues high and diagnostic of death. Long, low intake, which may occur in the field, may kill by causing edema and related phenomena; signs vary between individuals and between species. Under these circumstances, mortality resulting from PCB's would not necessarily be accompanied by high residues of PCB's in brains, and for these birds no good diagnostic technique is available, for the signs are nonspecific and the residue levels vary greatly.

*Patuxent Wildlife Research Center, U.S. Fish and Wildlife Service, Laurel, Maryland.

PCB's were implicated in a dieoff of guilemots (*Uria aalge*) which occurred in the Irish Sea region in 1969 (ref. 8). The body load of PCB's in birds that died was about twice that in healthy birds collected in the same general region. Moreover, livers of shot birds contained only 0.9 percent of the total body burden compared to 22.5 percent for birds found dead. PCB residues in brains were not determined.

Uptake and Loss Rates

PCB's are readily taken up by animal tissues, and loss rates are reasonably slow. Like DDT and other lipid-soluble pesticides, PCB's accumulate in adipose tissue and have been shown to move out of adipose tissue during lipid mobilization. In experimentally dosed birds, highest PCB levels accumulated in adipose tissue, followed by kidney, liver, brain, muscle, and blood (refs. 3,9).

In experimentally dosed grackles (*Quiscalus quiscula*), half of the PCB's were gone from the tissues in about 1 month, compared to an estimated 6 months for DDE (ref. 4). Continued loss of PCB's after the first month, however, was exceedingly slow and there was no significant further loss occurring during the next 12 weeks. After 32 weeks, about 15 percent of the original burden of PCB's remained in the body.

PCB's are readily excreted in the egg. PCB levels in the eggs of double-crested cormorants (*Phalacrocorax auritus*) reflected carcass levels, but this relationship did not hold for white pelicans (*Pelacanus erythrorhynchos*) (ref. 10). In ringdoves (*Streptopelia risoria*), Aroclor 1254 fed at 10 ppm reached an equilibrium level of 17 ppm wet weight in the eggs; this was about 2 percent of the level found in the fat of the adult birds (ref. 9).

PCB's stored in the adipose tissue are readily mobilized during periods of stress that cause loss of body weight. Alternate starving and feeding of pheasants dosed with PCB's increased the toxicity of the PCB's (ref. 11). Birds that died from starvation had mobilized their fat reserves and had considerably higher PCB's in the brain and other tissues than did PCB-treated birds that were not starved. Similarly, increased levels of PCB in muscle, brain, and liver of PCB-treated ringdoves followed depletion of fat reserves by starvation. At death, PCB concentration in the brains of starved birds had increased 56-fold (ref. 9).

Reproductive Effects

Effects of PCB's on reproduction are difficult to evaluate but are apt to have the most serious impact on populations. Few data are available for wild species, so work done with chickens must be relied upon. Numerous good studies have shown that chickens are sensitive to PCB's. The chief reproductive effects in chickens are lowered egg production and reduced hatchability. Deformities in chicks are common, and growth of young may be depressed. Survival of young sometimes is affected. Feed consumption, adult survival and body weight, eggshell characteristics, and gametogenesis usually are not affected. Most embryo mortality occurs during the latter stages of incubation, although this may depend upon the PCB residue in the egg. Many chicks may die after pipping. The most severe effects are produced by Aroclors in the middle of the range--from 1232 through 1254.

Chickens fed 2 ppm of several Aroclors in the diet for 9 weeks were not adversely affected (ref. 12). Others fed 20 ppm were affected differently by the different types of Aroclors. Aroclors 1221 and 1268 did not affect egg production or hatchability. Aroclor 1248 produced the most severe effects, causing some mortality of adults and nearly eliminating hatching of eggs. Aroclor 1242 produced effects that were very nearly as severe. Aroclor 1232 produced less effect.

The lack of effects of 2 ppm of dietary PCB on chicken reproduction was supported by a 39-week feeding test with Aroclor 1254 (ref. 13). Five ppm of 1254 reduced egg production erratically over the 39-week period. With 50 ppm, however, mortality began, and dosage was stopped after 14 weeks. Egg production fell sharply. Hatchability dropped nearly to zero; residues in eggs were then about 25 to 50 ppm. As residues in eggs dropped after 6 weeks of clean food, hatchability rose. Residues over 10 to 15 ppm in eggs were accompanied by heavy embryotoxicity, but those below 5 ppm showed no effect. At the start of the 50-ppm dosage, deaths of embryos came late in development, but as residues built up deaths came progressively earlier.

The effects on reproduction were further investigated in another study in which chickens were given 50 ppm Aroclor 1254 in water for 6 weeks, and then were given untreated water for 20 weeks (ref. 14). Hatchability dropped to zero within 3 weeks after dosage began and stayed nearly at zero through 8 weeks of clean water, then rose to approximately normal levels after 16 weeks of untreated water. The amount of embryonic mortality associated with a given amount of PCB residue in the egg became higher as the study progressed. Thus, 50 percent mortality of embryos was correlated with 50 ppm of PCB in the yolk after 1.6 weeks of dosage, but with 10 ppm after 18.7 weeks, a fivefold difference. The

greatest toxicity per unit of PCB came after 11 weeks of untreated water. Late in the study, 6 to 8 ppm of PCB in yolks was correlated with 14 to 36 percent mortality of embryos This would represent about 3.6 ppm in the whole egg. The authors concluded that the increase of toxicity with time was caused by the accumulation of some persistent isomer or homolog of 1254 or by a metabolite.

In another study with chickens, egg production was reduced by 10 percent and hatchability by 44 percent when eggs from hens dosed with Aroclor 1248 contained only 3 ppm (ref. 15). When levels in the eggs reached 4.5 ppm, production was further reduced and hatchability was almost completely eliminated.

PCB residues in eggs of wild birds have often equalled or exceeded the levels known to have caused reproductive problems in chickens. For a few examples, 11 eggs of bald eagles from the United States contained from 2.2 to 27.7 ppm of PCB's with a median of 9.7 ppm (ref. 16). PCB residues in osprey (*Pandion haliaetus*) eggs collected in Connecticut in 1968 and 1969 varied from 3.6 to 51 ppm (ref. 17) and brown pelican (*Pelecanus occidentalis*) eggs from the Eastern United States contained from 1.9 to 36.5 ppm PCB's, with many readings over 5 ppm (ref. 18). Species differences in response must be considered in the interpretation of the effects of these levels upon reproduction of wild birds. The number of species that may respond like chickens is not known.

PCB's caused deformities in chicks from hens dosed with 50 ppm Aroclor 1254 (ref. 19). Many deformities appeared when residues in yolks were 10 ppm or more. In another study in which hens were dosed with 20 ppm of Aroclors 1232, 1242, 1248, or 1254, 34 percent of 843 embryos that died during incubation showed signs of abnormal development (ref. 20). PCB's or related chemicals were suspected of causing deformities in a small number of young in a tern colony in which PCB residues were high (ref. 21).

Other species of birds may be less sensitive to PCB's than chickens. Mallards showed no decline in reproductive success during 2 years of dosage with 25 ppm of Aroclor 1254 (ref. 1). Similar results have been reported for coturnix quail (refs. 15,22) and bobwhite quail (ref. 1). Pheasants given oral doses of Aroclor 1254 showed depressed egg production and hatchability, suggesting a response similar to that of chickens (ref. 23).

Decreases in eggshell thickness and associated declines in populations of certain species of wild birds have been linked with elevated levels of DDE in their eggs (for review see ref. 24). The effect of DDE upon eggshell thickness has been demonstrated experimentally with several species (ref. 24). Recent experimental studies with PCB's, however, have failed to demonstrate that low dietary dosages cause significant eggshell thinning in mallards (ref. 1), bobwhite quail (ref. 1), pheasants (ref. 23), ringdoves (ref. 25), or chickens (refs. 12,15,26,27). In earlier tests, chickens fed Aroclor 1242 at 10 ppm or 100 ppm and Aroclor 1254 at 100 ppm laid eggs with thinner shells than did controls (ref. 28). Coturnix quail hens given 10 ppm Aroclor 1242 in the feed for 40 days produced eggs with shells 5 percent thinner than did hens given untreated feed (ref. 29). Similarly, 50 ppm of Aroclor 1254 in the diet produced a small decrease in shell thickness and an increase in the percentage of cracked eggs of coturnix quail (ref. 22). No tests of eggshell thinning have been made with birds of prey or other critical species.

Statistical evaluation of the role that different chemicals may play in thinning the eggshells of brown pelicans in the field has shown that DDE residues correlate much better with shell thinning than do residues of PCB's (ref. 18). Similar relationships have been shown for other species (ref. 30).

Effect on Chromosomes

Few cytogenetic studies have been conducted to determine effects of PCB's on chromosome structure. In one study, no chromosomal aberrations were detected in chicken embryos following injection of Aroclor 1242 into the egg to levels of 10 and 20 ppm (ref. 31). At these levels mortality of embryos was high. In another study, embryos from ringdoves treated with Aroclor 1254 at 10 ppm had a higher frequency of chromosome aberrations than did controls (ref. 32).

Chromosomal aberrations may contribute to the abnormalities of developing embryos observed in some studies, or perhaps to a decreased reproductive success during successive generations of exposure to PCB's. When ringdoves were exposed to 10 ppm Aroclor 1254 during two generations, the reproductive success of the first generation was normal, but the success of the second generation was greatly reduced (ref. 32).

Effects on Behavior

Alterations in behavior may bring about depressed survival and reproductive success of wild species. Experimental studies have shown that PCB's may alter behavior. Caged European robins (*Erithacus rubecula*) fed PCB's showed increased migratory activity compared with untreated controls (ref. 33). PCB's caused a similar tendency in redstarts (*Phoenicurus phoenicurus*) (ref.

34). Behavior, on a visual cliff, of pheasant chicks hatched from hens given 50 mg Aroclor 1254 weekly was significantly different from controls or those receiving 12.5 mg (ref. 23); more chicks from the 50-mg group jumped to the visually deep side or made no choice of sides than chicks from other groups. The avoidance response to a moving silhouette of coturnix quail chicks fed 200 ppm of Aroclor 1254 was greatly reduced (ref. 35); there was no significant recovery in response after the birds were again given clean feed, suggesting an effect upon the maturing central nervous system.

Embryonic mortality of ringdoves fed 10 ppm Aroclor 1254 was higher when the eggs were incubated by the parent birds than when they were incubated artificially (ref. 36). Egg temperatures suggested that the increased mortality was due to decreased parental attentiveness.

EFFECTS ON MAMMALS

Certain species of mammals appear to be especially sensitive to ingestion of low levels of PCB's. An extreme example was provided by the declining reproductive success and increasing mortality of mink that was observed by commercial mink ranchers in the Great Lakes region in the 1960's. The problems developed when mink ranchers began to use coho salmon from the Great Lakes in mink rations (ref. 37). Other fish from Lake Michigan had similar effects, but coho from the Pacific coast caused no trouble. Feeding trials were conducted with several contaminants that had been identified in the fish (ref. 38). Neither DDT nor dieldrin caused these effects in mink, even at levels far higher than those in the fish. In the first test, a mixture of PCB's at 30 ppm in the diet killed all the adult mink. With dietary dosages of 5 and 10 ppm of Aroclor 1254, no reproduction occurred. At 10 ppm, five of the six adults died. In further tests, 1 ppm of PCB in the diet reduced reproductive success. More drastic results were observed when the mink were fed meat from cows that had been dosed with Aroclor 1254 (ref. 39). When the concentration of PCB in the diet was 3.57 ppm, there was no reproduction and all breeders died. When the concentration was 0.64 ppm, some adults died and no surviving young were produced.

High levels of PCB's and organochlorine insecticides have been found in marine mammals and a link with premature births in some species has been suggested. Premature pups from California sea lions (*Zalophus californicus*) contained higher levels of these compounds in the blubber, liver, and brain than did full-term pups (ref. 40).

Recent studies have shown that certain species of bats are sensitive to PCB's. In big-brown bats (*Eptesicus fuscus*), PCB's crossed the placenta 2 to 3 times more readily than DDE (ref. 41). Concentrations of PCB's were significantly greater in litters that included dead young than in litters in which both young were born alive. In further studies, big-brown bats fed 250 ppm DDE in the diet gained weight and none died, but individuals fed only 10 ppm Aroclor 1254 gained weight more slowly and some died (ref. 42).

PCB's have been shown to be toxic to some nonhuman primates over a wide dose range. Adult rhesus monkeys developed signs of PCB intoxication within 1 to 2 months at doses as low as 2.5 and 5.0 ppm of Aroclor 1248 in the diet (ref. 43); these levels also caused a marked decline in reproductive success.

CONCLUSIONS

The effects of PCB's upon wild populations are difficult to evaluate and many important questions remain to be answered. Residues in wild species may be derived from different commercial mixtures that have undergone metabolic changes for varying time periods before reaching the target organism. Residues of PCB's in wild birds often are at levels known to have caused reproductive impairment in chickens. Nevertheless, species differences must be considered and it is not known how many species respond in the same manner as chickens. The great sensitivity of mink raises the question of how many other mammals may be equally sensitive to reproductive impairment or mortality.

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